PALM INTRANET

Day: Wednesday

Date: 6/23/2004 Time: 11:00:49

## **Inventor Information for 10/043658**

Inventor Name	City	State/Country
OLSON, ERIC N.	DALLAS	TEXAS
Appln Info Contents Petition Info	Atty/Agent Info	Continuity Data Foreign Data
Search Another: Application#	0	or Patent# Search
PCT/ /	Search	G PUBS #
Attorney Docket # [		Search
Bar Code #	Search	

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

<b>(**</b>	PAL	MIN	TRAI	VET
------------	-----	-----	------	-----

Day: Wednesday

Date: 6/23/2004 Time: 11:00:55

## **Continuity Information for 10/043658**

**Parent Data** 

10043658

is a continuation of <u>09438075</u>

Which Claims Priority from Provisional Application 60107755

Which Claims Priority from Provisional Application 60108083

## Child Data No Child Data

Appin Info Contents Petition Info Atty/Agent	Info Continuity Data Inven
Search Another: Application#	or Patent# Search
PCT / Search	or PG PUBS #
Attorney Docket #	Search
Bar Code # Se	parch.

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page







**PMC** PubMed Nucleotide Protein Genome Structure OMIM Journals Search PubMed Go Clear for mef2 heart hypertrophy Limits Preview/Index Clipboard Details History About Entrez Show: 20 Display Summary Sort Send to Items 1-7 of 7 One pag **Text Version** 1: Czubryt MP, Olson EN. Related Articles, Link: Entrez PubMed Balancing contractility and energy production: the role of myocyte enhancer Overview factor 2 (MEF2) in cardiac hypertrophy. Help | FAQ Recent Prog Horm Res. 2004;59:105-24. Review. Tutorial New/Noteworthy PMID: 14749499 [PubMed - indexed for MEDLINE] E-Utilities 2: Zhang CL, McKinsey TA, Chang S, Antos CL, Hill JA, Olson EN. Related Articles, Links PubMed Services Class II histone deacetylases act as signal-responsive repressors of cardiac Journals Database hypertrophy. MeSH Database Single Citation Matcher Cell. 2002 Aug 23;110(4):479-88. **Batch Citation Matcher** PMID: 12202037 [PubMed - indexed for MEDLINE] Clinical Queries 13: Liu ZP, Nakagawa O, Nakagawa M, Yanagisawa H, Passier R, LinkOut Related Articles, Links Cubby Richardson JA, Srivastava D, Olson EN. CHAMP, a novel cardiac-specific helicase regulated by MEF2C. Related Resources Dev Biol. 2001 Jun 15;234(2):497-509. **Order Documents** PMID: 11397016 [PubMed - indexed for MEDLINE] **NLM Gateway TOXNET** Related Articles, Links 4: Han J, Molkentin JD. Consumer Health Clinical Alerts Regulation of MEF2 by p38 MAPK and its implication in cardiomyocyte ClinicalTrials.gov biology. PubMed Central Trends Cardiovasc Med. 2000 Jan; 10(1):19-22. Review. PMID: 11150724 [PubMed - indexed for MEDLINE] Related Articles, Link 5: Olson EN, Williams RS. Remodeling muscles with calcineurin. Bioessays. 2000 Jun;22(6):510-9. Review. Erratum in: Bioessays 2000 Nov;22(11):1049. PMID: 10842305 [PubMed - indexed for MEDLINE] 6: Passier R, Zeng H, Frey N, Naya FJ, Nicol RL, McKinsey TA, Related Articles, Link: Overbeek P. Richardson JA, Grant SR, Olson EN. CaM kinase signaling induces cardiac hypertrophy and activates the MEF2 transcription factor in vivo. J Clin Invest. 2000 May;105(10):1395-406. PMID: 10811847 [PubMed - indexed for MEDLINE] 7: Kolodziejczyk SM, Wang L, Balazsi K, DeRepentigny Y, Kothary Related Articles, Link: R, Megeney LA. MEF2 is upregulated during cardiac hypertrophy and is required for normal post-natal growth of the myocardium. Curr Biol. 1999 Oct 21;9(20):1203-6. PMID: 10531040 [PubMed - indexed for MEDLINE] Show: 20 Sort Send to Text Display Summary

Items 1-7 of 7

One page

Write to the Help Desk

NCBI | NLM | NIH

Department of Health & Human Services

Privacy Statement | Freedom of Information Act | Disclaimer

b e

Jun 7 2004 18:11:

 $h \hspace{1cm} cb \hspace{1cm} h \hspace{1cm} g \hspace{1cm} e \hspace{1cm} e \hspace{1cm} fcg \hspace{1cm} e \hspace{1cm} ch$ 

В







Send to

**PMC** Journals Nucleotide Protein Genome Structure OMIM Entrez PubMed Search PubMed Go Clear v for Limits Preview/Index History Clipboard Details

About Entrez

**Text Version** 

Entrez PubMed Overview Help | FAQ Tutorial New/Noteworthy E-Utilities

PubMed Services
Journals Database
MeSH Database
Single Citation Matcher
Batch Citation Matcher
Clinical Queries
LinkOut
Cubby

Related Resources Order Documents NLM Gateway TOXNET Consumer Health Clinical Alerts ClinicalTrials.gov PubMed Central

h

cb

☐ 1: Recent Prog Horm Res. 2004;59:105-24.

Related Articles, Linl

Text

Full text article at rphr.endojournals.org

Abstract

Display :

Balancing contractility and energy production: the role of myocyte enhancer factor 2 (MEF2) in cardiac hypertrophy.

Sort

Show: 20

Czubryt MP, Olson EN.

University of Texas Southwestern Medical Center at Dallas, Dallas, Texas 75390-9148, USA.

Cardiac hypertrophy -- that is, enlargement of the heart resulting from increased myocyte size -- is observed with many forms of human heart disease. It may arise secondary to an insult, such as infarct or chronic hypertension, or may occur as a consequence of a genetic defect, such as in hypertrophic cardiomyopathy. Traditionally, it has been widely believed that hypertrophy occurred as an adaptive response to normalize increased wall stress due to disease. Recently, however, it has been observed that while hypertrophy initially appears to improve the function of the heart following insult, over time, it frequently leads to a decompensated state, characterized by fibrosis and chamber dilation, resulting in overt heart failure. Hypertrophy also occurs during fetal development, immediately after birth, and in trained athletes; however, it does not lead to decompensation in these states. Experiments over the last 15 years have implicated similar signaling pathways in both pathological and physiological hypertrophic responses. Recently, important differences have been demonstrated that might hold the key to the development of effective new treatments for human diseases. This chapter focuses on how these hypertrophic responses differ from one another phenotypically and discusses how inefficient or impaired energy metabolism in the heart may contribute to the development of pathological responses. We also discuss recent evidence that the myocyte enhancer factor 2 (MEF2) transcription factor family, which previously has been shown to be important in cardiac development and hypertrophy, may have a role in regulation of cardiac energy metabolism.

**Publication Types:** 

- Review
- · Review, Tutorial

PMID: 14749499 [PubMed - indexed for MEDLINE]

cb

h

Display Abstract Show: 20 Sort Send to Text

Write to the Help Desk

NCBI | NLM | NIH

Department of Health & Human Services

Privacy Statement | Freedom of Information Act | Disclaimer

Jun 7 2004 18:11: